ABSTRACT OF THE DISCLOSURE

The present invention provides transgenic mice deficient in corticotropin releasing factor receptor 2 (CRFR2). Mice deficient for CRFR1 exhibit decreased anxiety-like behavior and a decreased stress response. In contrast, CRFR2 null mutant mice are hypersensitive to stress and display increased anxiety-like behavior. These mice are useful for the study of anxiety, depression, and the physiology of the HPA axis. CRFR2 null mutant mice also exhibit increased angiogenesis in all tissues examined. Thus, CRFR2 antagonists may be used to stimulate angiogenesis for the treatment of various conditions. In contrast, CRFR2 agonists may be used to inhibit angiogenesis. A combination of urocortin and bFGF was observed to stimulate rapid hair growth.